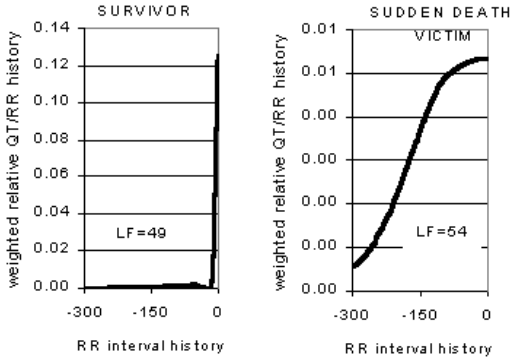
patients who died during follow-up in both A and P treated patients separately.
Results: In both A and P arm, patients who died had significantly greater LF-values than survivors (A 47.7± 11.0 vs 45.7± 7.5, p=0.03; P 53.6± 9.4 vs 48.8± 7.4, p<0.005). In both survivors and those who died, LF-values on A were significantly smaller than on placebo (p<0.005 in both comparisons).
Conclusions: In high-risk post-MI patients, the QT interval adaptation to heart rate was significantly slower than in survivors. An increase in adaptation speed on A might be an explanation for the antiarrhythmic efficacy of the drug.



1015-222 **Electrocardiographic Progression of Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy**

Chandra Bomma, Jonathan Piccini, Darshan Dalal, Daniyal Ahmed, Harikrishna Tandri, Khurram Nasir, Crystal Tichnell, Cynthia James, Julie Rutberg, Hugh Calkins, Johns Hopkins University School of Medicine, Baltimore, MD

Back Ground: Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) is a genetic cardiomyopathy characterized by fatty fibrous replacement of right ventricle (RV). Symptoms vary from ventricular arrhythmias to sudden cardiac death. The diagnosis of ARVD/C is based on International Task Force criteria (TFC). Although ARVD/C is considered to be a progressive condition, few data are available concerning the rate and extent of progression of this condition over time. The purpose of this study was to determine whether serial electrocardiographic (ECG) evaluations demonstrate evidence of progression of ARVD/C.
Methods and Results: The study population included (32) individuals (13 males; mean age 36±14 years) who were diagnosed with ARVD/C using the TFC. We analyzed the 12 lead ECG at two time points, separated by a median of 40 months. Progression on ECG was defined as new T-wave inversion beyond the extent of T-wave inversion at baseline, QRS progression of greater than 10 milliseconds duration over QRS duration at baseline, development of a new bundle branch block (BBB), and/or the appearance of a new Epsilon wave. Overall, ECG progression was observed in 20(62.5%) patients. T-wave progression was seen in 12(37.5%), QRS duration progression was seen in 4(12.5%) patients and a new BBB was seen in 4(12.5%) patients. Epsilon wave was seen in one patient and no new epsilon wave is seen in any of the patients.
Conclusion: The results of this study demonstrate that more than 50% of patients with ARVD/C demonstrate evidence of progression of the disease by ECG criteria. Further research is needed to determine what behavioral, clinical, and genetic factors best predict disease progression.

1015-223 **T Wave Morphology Abnormalities Are Associated With Clinical Outcome in Hypertrophic Cardiomyopathy**

Yi Gang, Katerina Hnatkova, Juan Gimeno, Marek Malik, St. George's Hospital Medical School, London, United Kingdom

Background: T-wave morphology (TWM) analysis quantifies repolarization abnormalities. This study evaluated the relation of TWM to risk factors (RF: a family history of sudden cardiac death (SD), a history of VF, recurrent syncope, non-sustained VT, an abnormal blood pressure response on exercise, maximum left ventricular wall thickness ≥30 mm) for SD and clinical outcome in patients (pts) with hypertrophic cardiomyopathy (HC).
Methods: Digitised 12-lead ECGs were recorded from 157 HC pts (107 men, age 43±15 yrs) using a MACVU electrocardiograph (GE Medical Systems, US) with a sampling rate of 500 Hz. Study pts were followed up for 60±44 months. Analysis of the digital ECG recordings was performed in a fully automatic manner with custom-developed software implemented on a personal computer. Three TWM indices were calculated: complexity ratio (CR), the total cosine R-to-T (TCRT), and relative T-wave residuum (TWR%).
Results: TWR% and CR were significantly increased in pts with any RF (0.14±0.02, 0.25±0.02) or with ≥2 RF (0.11±0.01, 0.23±0.02) compared with pts without any RF (0.06±0.01, 0.20±0.01) (p<0.05, p<0.01), respectively. TCRT values were significantly lower in pts with cardiac event (cardiac death, VF, ICD discharge) or cardiac death compared with event-free survivors (table). A weak and significant correlation was found between TCRT and maximum left ventricular wall thickness (r=-0.23, p=0.004).
Conclusion: Abnormalities in TWM are associated with RF or clinical outcome in HC pts.

TCRT (mean±sem)	positive	negative	P value
Cardiac events (n=14)	-0.67±0.09	—0.43±0.04	0.030
Cardiac death (n=9)	-0.71±0.12	—0.43±0.04	0.035

1015-224 **ST-Segment Elevation in Lead AVR in Acute Myocardial Infarction-Clue to Circumflex Coronary Artery Origin for Inferior/Posterior Myocardial Infarction**

Chandana Weerasinghe, Giselle Smith, Mano Senaratne, Grey Nuns Hospital-University of Alberta, Edmonton, AB, Canada

Background: Inferior/posterior acute myocardial infarctions(AMI) can result from occlusion of the right or circumflex coronary arteries. Right pre-cordial and posterior leads are often useful in this differentiation. The present study evaluated the utility of ST-segment elevation (STE) in lead aVR in this differentiation.
Methods: ST-segment change (approximated to the nearest 0.5mm once a minimum of 0.5mm of deviation was identified) was measured with the naked eye on the presenting electrocardiogram(EKG) in 561 consecutive pts with AMI (M=408, F=153; age: mean 64.8±0.6 yrs SEM). All the EKGs were evaluated by one individual who was blinded to the rest of the clinical and investigational data. In all leads STE of 0.5 mm or more was considered significant. All data were collected prospectively.
Results: 137 patients (24.4%) demonstrated STE in aVR with 17.3%, 26.4%, 38.7%, and 42.0% incidences in inferior, antero-septal, posterior, and lateral AMI's respectively (p<0.001). Those with STE in aVR had higher magnitudes of ST-segment depression (STD) in leads I, II, V1, V2, V4, aVL and higher magnitudes of STE in leads II, III, aVF, V5, V6 (p<0.01). The presence of STE in the right pre-cordial leads was not significantly associated with STE aVR (p>0.05). In patients in whom an angiogram was done within the hospital stay (138 pts=24.6%) STE in aVR was present in 15.4%, 30.8%, 52.4%, and 60.0% of patients with the culprit lesion in the right coronary, left anterior descending, circumflex, and obtuse marginal arteries respectively (p=0.003). STE in aVR appeared to be associated with significantly lower creatine kinase peaks (1214±123 u/l vs. 1753±152 u/l, p=0.05) with a trend towards a lower in-hospital mortality (2.9% vs. 5.9%) and one-year coronary event rates (18.7% vs. 28.1%, p=0.09).
Conclusions: STE in aVR is more often associated with inferior and/or posterior AMI's caused by a circumflex coronary artery occlusion (as opposed to a right coronary artery occlusion). Thus, it may serve as a useful additional marker to help in this differentiation.

POSTER SESSION

1034

Basic Mechanisms of Atrial and Ventricular Fibrillation

Sunday, March 07, 2004, Noon-2:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1034-207 **Mechanism for Rate Slowing by Anterior Atrioventricular Node Modification During Atrial Fibrillation: Partial Damage of the Compact Node Rather Than Fast Pathway Ablation**

Youhua Zhang, Saroja Bharati, Rabi Sulayman, Todor N. Mazgalev, The Cleveland Clinic Foundation, Cleveland, OH, Hope Children's Hospital, Oak Lawn, IL

Background: We have demonstrated that selective fast pathway (FP) ablation has no effect on ventricular rate (VR) in atrial fibrillation (AF). This appears to contradict reports of VR slowing in about 1/3 of patients using anterior approach. We hypothesized that such clinical outcome results from inadvertent damage of the compact atrioventricular node (AVN).
Methods: His electrogram alternans (Circ 2003;107:1059) was used to monitor the dual pathway conduction during AF in 12 rabbits. The FP conduction resulted in low amplitude inferior His electrogram (IHE), while slow pathway (SP) - in high IHE. After successful selective FP ablation, further lesions were applied progressively closer to the compact AVN. The ablation sites were studied by detailed morphological sectioning.
Results: Both FP and SP conducted beats (Figure A, low and high IHE) were present during AF. Selective FP ablation revealed only-SP conduction (B, high IHE), but didn't change VR (298±18 vs 301±14 bpm, P>0.05). Ablations closer to AVN resulted in SP-rate slowing (C) and ultimately in AVN block (not shown). Morphological evidence indicated that VR slowing occurred only when lesions extended into the compact AVN domain resulting in a non-selective, FP plus SP, modification.
Conclusion: Selective FP ablation has no effect on VR in AF. Slower VR could be